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THE DISAPPEARANCE OF THE ACID MUCOPOLYSACCHARIDE COATING IN BLOOD VESSELS EXPOSED TO OXYGEN, RADIATION AND IN OLD AGE AND POSSIBLE IMPLICATIONS

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The blood vessels are known to be involved in radiation pathology, both the fragility and permeability being altered. Delayed radiation response has been shown to involve a loss of the blood supply to necrotic areas. Since the structure of the mucopolysaccharide (MPS) coating on the luminal wall is heparin-like in nature and heparin has known anti-coagulant properties, the observed increase in clotting in older persons suggests possible alteration of the acid MPS coating.

Rats were treated in several ways to examine the effect of various factors on the MPS coating. Young rats were whole-body irradiated with 2400r X-rays and sacrificed 24 and 48 hours later. Another group of rats were exposed to 100% oxygen at one atmosphere for periods up to 72 hours. A third group of rats 2 years old or more were examined for the effects of age. The animals were perfused with 7% glutaraldehyde, followed by Luft's method of staining with ruthenium red. The heart and trachea were embedded in Epon/Araldite, sectioned and viewed, unstained (Fig. 1), at 40KV using a 20 micron objective aperture.

Both the irradiated rats and those exposed to 100% oxygen lost the MPS blood vessel coating (Figs. 2, 3). The blood vessels were edematous and the pinocytotic vesicles had increased in size (Fig. 4). The older rats showed a decrease in the MPS coating with increasing age. Otherwise their capillaries were generally normal in appearance.

Radiation and oxygen depolymerize mucopolysaccharides. The sulfate and carboxyl groups in the acid mucopolysaccharides bind calcium and calcium binding is the origin of anti-coagulant properties of the acid mucopolysaccharide. The loss of a MPS endothelial coating in the lumen through depolymerization, or the loss of the cells ability to produce the MPS coating would have certain implications in regard to blood flow. The loss of such a barrier between the blood and the endothelial membrane suggests surface contact with the blood elements and an increased chance of coagulation, when other conditions are just right. For example, blood stasis in a denuded area might bring about a clot. Vascular edema is reported to be associated with delayed radionecrosis and fibrin threads have been reported in necrotic areas. Depolymerization of the MPS would increase the number of molecules present and increase the osmotic pressure. The consequences of such depolymerization by oxygen and radiation offer one explanation for the observed edema and the loss of the MPS coating. This in turn offers an explanation for loss of blood flow in areas where delayed radiation effects occur. Although the older rats which lacked MPS coatings did not have edematous vessels, loss of MPS may still be an age related factor and occur as a result of loss of production rather than depolymerization of this coating material.

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Fig. 1 Blood vessel wall stained with ruthenium red 100,000X

Fig. 2 Rat heart irradiated with 2400r X-rays 8,500X

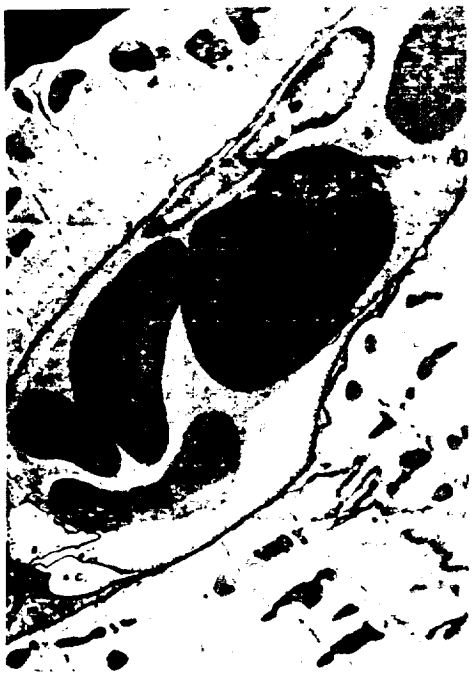

Fig. 3 Rat heart exposed to 100% O_2 for 72 hours 7000X

Fig. 4 Rat heart irradiated with 2400r X-rays 30,000X